(FILE 'USPAT' ENTERED AT 16:14:31 ON 28 APR 1998)

L1	645	S	P53
L2	0	S	L1(P)MUTATION3
L3	160	S	L1(P)MUTATION#
L4	85	S	L3(P)(SEQUENC?)
L5	64	S	L4 AND (DIAGNOS? OR PROGNOS?)
L6	14	S	L4(P)(DIAGNOS? OR PROGNOS?)
L7	32	S	L3(P)BREAST
L8	11	S	L4(P)BREAST

Set	Items	Description
S1	91619	P53
S2	33749	S1(15N)(MUTATION?)
S3	5706	S2(15N)(SEQUENC?)
S4	217	S3(15N)(DIAGNOS? OR PROGNOS?)
S5	21	S4(15N)(DNA(W)BINDING OR TRANSACTIVATION OR CONSERVED)
S6	11	RD (unique items)
S7	53	S4 AND BREAST
S8	26	RD (unique items)
S9	153	S4 NOT (S5 OR S7)
S10	70	RD (unique items)
S11	2578	S2(15N)(BREAST)
S12	506	S11(15N)(DIAGNOS? OR PROGNOS?)
S13	145	S12/1985:1994
S14	86	RD (unique items)
S15	86	S14 NOT S10
S16	290	AU="BYWATER M":AU="BYWATER MT"
S17	1	S16 AND P53
S18	806	AU="LINDSTROM P":AU="LINDSTROM PW"
S19	1	S18 AND P53
S20	332	AU="INGANAS M":AU="INGANAS, MATS"
S21	30	S20 AND P53

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8/7/9 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09468323 98196537
Prognostic value of P53 gene mutations in a large series of node-negative **breast** cancer patients.
Falette N; Paperin MP; Treilleux I; Gratadour AC; Peloux N; Mignotte H; Tooke N; Lofman E; Inganas M; Bremond A; Ozturk M; Puisieux A
Unite d'Oncologie Moleculaire, Unite INSERM U453, Centre Leon Berard, Lyon, France.
Cancer Res (UNITED STATES) Apr 1 1998, 58 (7) p1451-5, ISSN 0008-5472 Journal Code: CNF
Languages: ENGLISH
Document type: JOURNAL ARTICLE
The most important subgroup of **breast** cancer patients for which reliable prognostic factors are needed are women without axillary lymph node involvement. Although overall, these patients have a good prognosis, it is known that 20-30% will experience a recurrence of the disease. To determine the **prognostic** significance of **P53** tumor suppressor gene **mutation**, specimens from 113 primary **breast** cancers were evaluated for the presence of **P53** alterations, as detected by cDNA **sequencing** of the entire coding **sequence** of the gene. The median follow-up for patients was 105 months. **P53** gene **mutation** was an independent **prognostic** marker of early relapse and death. Our results suggest that **P53** gene **mutations** could be an important factor to identify node-negative patients who have a poor **prognosis** in the absence of adjuvant therapy. Prospective studies should be designed to determine which therapy should be performed in this subgroup of patients.

8/7/10 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08536906 96165524
Mutation detection by highly sensitive methods indicates that p53 gene mutations in **breast** cancer can have important prognostic value.
Kovach JS; Hartmann A; Blaszyk H; Cunningham J; Schaid D; Sommer SS
Department of Oncology, Mayo Clinic, Rochester, MN 55905, USA.
Proc Natl Acad Sci U S A (UNITED STATES) Feb 6 1996, 93 (3) p1093-6, ISSN 0027-8424 Journal Code: PV3
Contract/Grant No.: R01-CA56881, CA, NCI; CA15086, CA, NCI
Languages: ENGLISH
Document type: JOURNAL ARTICLE
Human cancer cells with a mutated p53 tumor-suppressor gene have a selective growth advantage and may exhibit resistance to ionizing radiation and certain chemotherapeutic agents. To examine the prognostic value of mutations in the p53 gene, a cohort of 90 Midwestern Caucasian **breast** cancer patients were analyzed with methodology that detects virtually 100% of all mutations. The presence of a p53 gene mutation was by far the single most predictive indicator for recurrence and death (relative risks of 4.7 and 23.2, respectively). Direct detection of p53 mutations had substantially greater prognostic value than immunohistochemical detection of p53 overexpression. Analysis of p53 gene mutations may permit identification of a subset of **breast** cancer patients who, despite lack of conventional indicators of poor prognosis, are at high risk of

8/7/12 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

08218031 94235435
p53 immunohistochemical analysis in **breast** cancer with four monoclonal antibodies: comparison of staining and PCR-SSCP results.
Jacquemier J; Moles JP; Penault-Llorca F; Adelaide J; Torrente M; Viens P ; Birnbaum D; Theillet C
Departement d'Anatomo-Pathologie, Institut Paoli-Calmettes, Marseille, France.
Br J Cancer (SCOTLAND) May 1994, 69 (5) p846-52, ISSN 0007-0920
Journal Code: AV4
Languages: ENGLISH
Document type: JOURNAL ARTICLE
The expression of p53 protein was examined in a series of 136 primary **breast** carcinomas, 106 of which were analysed with a panel of four monoclonal antibodies (MAbs 1801, 240, DO7 and DO1). p53 expression was detected with at least one antibody in 40 tumours (38%), whereas only 15 tumours (14%) were positive with all four antibodies. Some variability in the immunostaining could be observed depending on the antibody used. This was noticeable both for the number of positive cells within a section and for the intensity of staining. We therefore selected a panel of 17 tumour sections (nine were highly positive, three with medium to low staining and five with low to negative staining), which we analysed by polymerase chain reaction-single-strand conformation polymorphism analysis (PCR-SSCP) for the presence of a p53 mutation at the molecular level. Mutations were identified in 15 cases. Therefore the proportion of p53-stained cells does not seem to be an exact representation of the number of cancer cells bearing a mutation within a tumour. A statistically significant correlation was observed between p53 expression, regardless of the number of positive antibodies, and grade III disease ($P < 0.0001$), oestrogen ($P < 0.0001$) or progesterone receptor negativity ($P = 0.0061$), increased Ki 67 index ($P = 0.0018$), epidermal growth factor receptor (EGFR) positivity ($P = 0.0076$) and aneuploidy ($P = 0.037$). No correlation was observed with tumour size or lymph node involvement. In univariate analysis p53 expression was not correlated with disease-free survival, in contrast to the classical prognostic parameters, which were statistically correlated. In this series p53 expression was not a marker of early recurrence.

8/7/13 (Item 5 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07759824 94282791
The role of TP53 in **breast** cancer development.
Eeles RA; Bartkova J; Lane DP; Bartek J
Section of Molecular Carcinogenesis, Institute of Cancer Research, Sutton, Surrey.
Cancer Surv (UNITED STATES) 1993, 18 p57-75, ISSN 0261-2429
Journal Code: CNG
Languages: ENGLISH
Document type: JOURNAL ARTICLE; REVIEW; REVIEW, ACADEMIC
The TP53 protein is proving to be central to cell cycle control after exposure to DNA damage, and every week a new feature of its role in the regulation of cell division is described. TP53 is the most commonly altered oncogene in human tumours and is involved in the development of both sporadic and some hereditary **breast** tumours. Although there is no

doubt that germline mutations in the TP53 gene carry a high risk of early onset **breast** cancer and the gene is mutated somatically in a proportion of preinvasive and invasive **breast** cancers, it is not usually the initiating genetic event in most **breast** tumours. It does, however, seem to be an independent prognostic factor for survival and could prove useful in clinical management of node negative **breast** cancer patients. There has been an explosion of reports about the function of the TP53 protein--in particular, it seems to have a central role in the monitoring of some types of DNA damage. This role may prove to be the most important aspect of its association with **breast** cancer development, both as a prognostic factor and as a handle for treatment. (115 Refs.)

8/7/14 (Item 1 from file: 357)
DIALOG(R)File 357:Derwent Biotechnology Abs

177800 DBA Accession No.: 95-04621
A new approach in automated DNA sequencing to analyze the p53 gene in a large number of **breast** cancer patients - diagnosis by mamma carcinoma automated DNA sequencing and large-scale polymerase chain reaction (conference abstract)
AUTHOR: Andell Y; Eriksson S; Inganas M; Norberg T; Sevigny P
CORPORATE AFFILIATE: Pharmacia-Biotech
CORPORATE SOURCE: Pharmacia Biotech AB, Uppsala, Sweden.
JOURNAL: Clin.Chem. (40, 12, 2337) 1994
ISSN: 0009-9147 CODEN: CLCHAU
CONFERENCE PROCEEDINGS: The Genetic Revolution, San Diego, California, 17-19 November, 1994.
LANGUAGE: English
ABSTRACT: Frozen mamma carcinoma tissue mRNA was converted into cDNA and a method for amplification of the p53 gene by the polymerase chain reaction (PCR) was developed after dividing the 1.2 Kbp of the coding region of the cDNA sequence in 4 overlapping fragments. Streptavidin coated combs allowed the capture of a biotinylated PCR fragment by carrying the comb form wells to wells, containing the appropriate blend of reagents. With these combs and wells, the complete sequencing process was performed using phage T7 DNA-polymerase (EC-2.7.7.49) starting from a fluoresceinated primer. The sequencing material was then loaded directly from the combs on a sequencer. The data obtained were then transferred into a dedicated software to reveal any deviation in the p53 sequence. This simplified method provided a turn-around time of these 20 to 30 pts in one day, starting from the frozen tumor to the generation of a complete analysis report. 17% Of the material showed mutations in p53; 84% of the mutations were point mutations, 1% were in-frame insertions, 3% were out-of-frame insertions, 8% were in-frame deletions and 4% were out of frame deletion. (1 ref)

8/7/15 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

120213913 CA: 120(17)213913h JOURNAL
Prognostic significance of TP53 alterations in breast carcinoma
AUTHOR(S): Andersen, T.I.; Holm, R.; Nesland, J.M.; Heimdal, K.R.; Ottestad, L.; Boerresen, A. L.
LOCATION: Dep. Genet., Norw. Radium Hosp., N-0310, Oslo, Norway
JOURNAL: Br. J. Cancer DATE: 1993 VOLUME: 68 NUMBER: 3 PAGES: 540-8
CODEN: BJCAAI ISSN: 0007-0920 LANGUAGE: English
SECTION:
CA214001 Mammalian Pathological Biochemistry
IDENTIFIERS: gene TP53 mutation breast carcinoma human, antigen p53 mutation breast carcinoma human
DESCRIPTORS:

Gene,animal, c-erbB2.
amplification of, human breast carcinoma, gene 3 mutations in relation to
Mammary gland,neoplasm, carcinoma...
gene TP53 mutations in, in human, prognostic significance of
Mutation,deletion... Mutation,point... Mutation,transition...
in gene TP53 in human breast carcinoma, prognostic significance of
Estrogens,receptors... Progestogens,receptors... Receptors,estrogen...
Receptors,progestogen...
in human breast carcinoma, gene TP53 mutations in relation to
Gene,animal, TP53... Phosphoproteins,tumor suppressor, p53...
mutations in, in human breast carcinoma, prognostic significance of
Deoxyribonucleic acid sequences...
of gene TP53 mutations, in human breast carcinoma, prognostic significance of
Protein sequences...
of tumor antigen p53 mutations, in human breast carcinoma, prognostic significance of
Cell nucleus...
tumor antigen p53 accumulation in, in human breast carcinoma, gene mutation in relation to
Genetic element,exon...
7, of gene TP53 in human breast carcinoma, prognostic significance of

10/7/17 (Item 17 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

11218454 BIOSIS Number: 97418454
Analysis of p53 gene mutations in acute myeloid leukemia
Trecca D; Longo L; Biondi A; Cro L; Calori R; Grignani F; Maiolo A T; Pelicci P G; Neri A
Serv. Ematol., Ospedale Maggiore, I.R.C.C.S., Via F. Sforza 35, 20122 Milano MI, ITL
American Journal of Hematology 46 (4). 1994. 304-309.
Full Journal Title: American Journal of Hematology
ISSN: 0361-8609
Language: ENGLISH
Print Number: Biological Abstracts Vol. 098 Iss. 007 Ref. 089266
We have previously reported the absence of mutations within exons 5-9 of the p53 gene in a panel of 30 cases of acute promyelocytic leukemia (APL), which represent the M3 FAB type of acute myeloid leukemia (AML). In the present report, we extend our analysis of p53 gene mutations to 70 cases of AML representative of the other FAB types of the disease, including M1 (16 cases), M2 (20 cases), M4 (17 cases), M5 (12 cases), and M6 (5 cases). DNAs were analyzed for p53 gene mutations in exons 5 to 9 by polymerase chain reaction (PCR), single-strand conformation polymorphism (SSCP), and direct sequencing of PCR amplified products. Mutant p53 alleles were detected in 5 of 70 cases; 1 case in exon 5, 2 cases in axon 6, and 2 cases in axon 7. The alterations of the p53 gene were represented by point mutation leading to an amino acid substitution in four cases, and deletion in the remaining case. In four of the five cases, direct **sequencing** indicated the loss of the normal **p53** allele; in the remaining case, two **mutations** were detected, presumably involving both **p53** alleles. Three cases showed **mutations** at **diagnosis**; in the remaining two, the **mutations** were observed in clinical relapse but not at **diagnosis** . Our results confirm the relatively low incidence of p53 mutations in AML and further support the evidence that p53 plays a role in leukemogenesis through a recessive mechanism (two-hit model) of inactivation of tumor suppressor activity.

10/7/18 (Item 18 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

11116124 BIOSIS Number: 97316124
Nonhereditary p53 mutations in T-cell acute lymphoblastic leukemia are associated with the relapse phase
Hsiao M H; Yu A L; Yeargin J; Ku D; Haas M
Dep. Biol., UCSD Cancer Cent., 0063, Univ. Calif. San Diego, 9500 Gilman Drive, La Jolla, CA 92093-0063, USA
Blood 83 (10). 1994. 2922-2930.
Full Journal Title: Blood
ISSN: 0006-4971
Language: ENGLISH
Print Number: Biological Abstracts Vol. 098 Iss. 002 Ref. 022636
We have previously reported that greater than 60% of human leukemic T-cell lines possess mutations in the p53 tumor suppressor gene. To determine whether T-cell acute lymphoblastic leukemia (T-ALL) patient samples possess p53 mutations, we screened peripheral blood- and bone marrow-derived leukemia samples, taken at **diagnosis** and at relapse,

for **p53 mutations** . Exons 4 through 9 and selected intron regions of the **p53** gene were analyzed using polymerase chain reaction-single-strand conformation polymorphism and direct **sequencing**. **p53 mutations** were found in 0 of 15 T-ALL **diagnosis** samples, as compared with 10 of 36 (28%) T-ALL relapse samples. To determine whether p53 mutations play a role in the recurrence (relapse) of T-ALL, two special groups of T-ALL patients were studied: (1) a group of 8 relapse patients whose disease was refractory to chemotherapeutic treatment, and (2) a group of 6 "paired" T-ALL cell samples from patients for whom we possess both diagnosis and relapse samples. Three of 8 relapsed patients (37.5%) whose disease was refractory to the reinduction of remission by chemotherapy possessed missense mutations of the p53 gene. All 3 cases had mutations in axon 5. Among the paired samples, 3 of 6 patients harbored p53 mutations at disease recurrence, but possessed only wild-type p53 alleles at diagnosis. One case had mutation on axon 4, 1 case in axon 5, and 1 case in axon 8 with loss of heterozygosity. These data clearly indicate that recurrence of T-ALL is associated with missense mutations in p53. Our results indicate that (1) mutations of p53 do occur in T-ALL in vivo, and such mutations are associated with the relapse phase of the disease; and (2) p53 mutation is involved in the progression of T-ALL. This conclusion is supported by our observation that the introduction of T-ALL-derived mutant p53 expression constructs into T-ALL cell lines further increases their growth rate in culture, enhances cell cloning in methylcellulose, and increases tumor formation in nude mice.

10/7/25 (Item 3 from file: 73)
DIALOG(R)File 73:EMBASE

8893650 EMBASE No: 93197509

Application of the p53 gene mutation pattern for differential diagnosis of primary versus metastatic lung carcinomas

Noguchi M.; Maezawa N.; Nakanishi Y.; Matsuno Y.; Shimosato Y.; Hirohashi S.

Pathology Division, National Cancer Center Research Inst, 1-1 Tsukiji 5-Chome, Chuo-ku, Tokyo 104 Japan

DIAGN. MOL. PATHOL. (USA) , 1993, 2/1 (29-35)
CODEN: DMPAE ISSN: 1052-9551
LANGUAGES: English SUMMARY LANGUAGES: English

The p53 gene mutation pattern was used as a diagnostic marker of multiple and second primary lung carcinomas. Nine cases of multiple carcinoma, which were suspected clinicopathologically to be double or triple primary carcinomas, were examined for p53 protein expression by immunohistochemistry and for genetic abnormality of the p53 gene by polymerase chain reaction (PCR)-single-strand conformation polymorphism (SSCP) analysis. Nine tumors from four cases gave a positive result upon both immunostaining for the p53 protein and PCR-SSCP analysis of the p53 gene. These nine tumors showed different mobility shifts between exons 5 and 8. The four cases were diagnosed genetically as multiple primary carcinomas. To confirm the results of PCR-SSCP analysis, five tumors from two cases that showed different mobility shifts were further analyzed for their nucleotide **sequences** , and it was found that all of them had point **mutations** at different codons in exons 5 and 8. These findings suggest that the **p53** gene **mutation** pattern is an effective marker for **diagnosis** of tumor multiplicity.

15/7/1 (Item 1 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

11809274 BIOSIS Number: 98409274
Detection of **p53** gene **mutations** and their protein overexpression in fine-needle biopsy specimens with false-negative **diagnoses** in **breast** cancer
Sato T; Okazaki A; Nakano M; Toda K; Okazaki M; Takahashi S; Sato N; Kikuchi K; Hirata K
Dep. Surg., Sapporo Med. Univ. Sch. Med., S-1, W-16, Chuo-ku, 060 Sapporo, Japan
Tumor Research 29 (0). 1994. 49-57.
Full Journal Title: Tumor Research
ISSN: 0041-4093
Language: ENGLISH
Print Number: Biological Abstracts Vol. 100 Iss. 006 Ref. 086866
To achieve a more accurate **diagnosis** in the first aspiration biopsy from **breast** tumor, **p53** gene **mutations** were detected by PCR-SSCP analysis in aspiration biopsy specimens taken from 26 patients with **breast** tumors. Of 26 aspirated cell specimens from **breast** tumors that were all initially **diagnosed** as being cytologically benign, 2 point **mutations** of the **p53** gene were detected and were subsequently proved to be cancer cells. Further, the p53 protein expression was also examined in the initial aspirated specimens and in the resected tumors that were rediagnosed as being malignant as a result of the second biopsy. Consequently, these p53 gene mutations did not appear to correlate with their protein overexpression in the aspiration biopsy specimens (all cases were negative), however, the specimens from 2 resected tumors that showed p53 gene mutations were positive. In addition, a positive ER level and DNA aneuploidy status were also found only in these two p53 gene mutation cases. Therefore, detection of p53 mutations in aspiration biopsy specimens may prove to be a useful method for detecting breast cancers.

15/7/2 (Item 2 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

11319274 BIOSIS Number: 97519274
P53 gene **mutations** in **breast** cancers in midwestern US women: Null as well as missense-type **mutations** are associated with poor **prognosis**
Saitoh S; Cunningham J; De Vries E M G; McGovern R M; Schroeder J J; Hartmann A; Blaszyk H; Wold L E; Schaid D; Sommer S S; Kovach J S
Dep. Oncology, Mayo Clinic and Mayo Foundation, Rochester, MN 55905, USA
Oncogene 9 (10). 1994. 2869-2875.
Full Journal Title: Oncogene
ISSN: 0950-9232
Language: ENGLISH
Print Number: Biological Abstracts Vol. 098 Iss. 011 Ref. 155013
We determined the pattern of mutations in exons 2-11 and adjacent intronic regions in breast cancers from Midwestern US white women. Twenty-one mutations were detected in 53 tumors (39.6%). Comparisons of the pattern of mutations within exons 5-9 showed that the frequency of missense mutations (44%) was lower in breast cancers of US Midwestern women than in most tumor types including breast cancers in other populations. Compared to

breast cancers reported in a Scottish population. US women had a high frequency of microdeletion mutations (P=0.006) and low frequency of G:C fwdarw T:A transversions (P=0.046). These findings suggest that environmental or endogenous factors contribute to p53 mutagenesis in mammary tissue to different extents among different populations. With a median follow-up of 19 months, the presence of a mutation was associated with shorter time to disease recurrence (P=0.05) and shorter survival (P=0.003). Putative dominant negative missense-type mutations (missense and in-frame microdeletions; P=0.001) and null mutations (hemizygous nonsense and frameshift mutations; P=0.007) were equally ominous. Thus, tumors with missense p53 mutations resulting in overexpression of a dysfunctional but otherwise intact protein have a clinical outcome similar to tumors with null mutations resulting in a truncated or garbled protein.

15/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

11102091 BIOSIS Number: 97302091
Poor **prognosis** of **p53** nuclear overexpression and **mutation** in inflammatory **breast** carcinoma
Riou G; Le M G; Travagli J P; Moll U M; Levine A J
Inst. Gustave Roussy, 94800 Villejuif, FRA
Proceedings of the American Association for Cancer Research Annual Meeting 35 (0). 1994. 220.
Full Journal Title: 85th Annual Meeting of the American Association for Cancer Research, San Francisco, California, USA, April 10-13, 1994. Proceedings of the American Association for Cancer Research Annual Meeting
ISSN: 0197-016X
Language: ENGLISH
Print Number: Biological Abstracts/RRM Vol. 046 Iss. 007 Ref. 107444

15/7/7 (Item 7 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

11041715 BIOSIS Number: 97241715
P53 gene mutations and steroid receptor status in breast cancer
Caleffi M; Teague M W; Jensen R A; Vnencak-Jones C L; Dupont W D; Parl F F
Dep. Pathology, Vanderbilt University, Nashville, TN 37232, USA
Cancer (Philadelphia) 73 (8). 1994. 2147-2156.
Full Journal Title: Cancer (Philadelphia)
ISSN: 0008-543X
Language: ENGLISH
Print Number: Biological Abstracts Vol. 097 Iss. 011 Ref. 158829
Background. There is increasing evidence linking development and progression of cancer to an accumulation of mutations at the genomic level. The most frequently mutated gene known to date in sporadic breast cancer appears to be the tumor suppressor gene p53. This study was designed to determine the frequency of p53 gene mutations in primary breast cancer, to correlate the presence of p53 mutations with established clinicopathologic parameters, including the estrogen receptor (ER) and progesterone receptor (PR) status, and to assess the **prognostic** significance of **p53 mutations** regarding patient survival. Methods. We examined the **p53** gene in genomic DNA samples from 192 primary **breast** cancers. Using denaturant gradient gel electrophoresis, the authors analyzed exons 5-9 in all tumors for mutations and performed DNA sequencing in 20 tumors to identify the exact nature of the p53 mutations. Results. p53 gene alterations were identified in 43 of the 192 tumors (22%), the majority localized in exons 5 and 6. DNA sequencing showed mostly missense mutations resulting from G or C substitutions. p53 mutations were found more often in tumors of younger women (P = 0.002), Afro-American women (P =

0.05), and in tumors lacking ER (P = 0.03), PR (P = 0.04), or both (P = 0.06). There were [redacted] significant correlations with [redacted]ily history, tumor size, histologic grade or type, nodal status, or disease stage. The overall survival rates showed no significant difference between patients with mutant and wild-type p53 tumors. The same was true when the comparison was limited to node-negative patients or patients with ER-positive or ER-negative tumors. Finally, there was no significant difference in survival between patients with tumors harboring mutations in exons 5 and 6 versus exons 7-9. Conclusions. The results of this and other studies demonstrate a consistent relationship between ER-positive tumors and wild-type p53 on one hand and ER-negative cancers and p53 mutations on the other. Our data do not support a significant prognostic role for p53 mutations in predicting survival.

15/7/8 (Item 8 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

10807268 BIOSIS Number: 97007268
Poor **prognosis** of **p53** gene **mutation** and nuclear overexpression of **p53** protein in inflammatory **breast** carcinoma
Riou G; Le M G; Travagli J P; Lelvine A J; Moll U M
Inst. Gustave Roussy, 94805 Villejuif Cedex, FRA
Journal of the National Cancer Institute (Bethesda) 85 (21). 1993. 1765-1767.
Full Journal Title: Journal of the National Cancer Institute (Bethesda)
ISSN: 0027-8874
Language: ENGLISH
Print Number: Biological Abstracts Vol. 097 Iss. 001 Ref. 006566

15/7/9 (Item 9 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

10136586 BIOSIS Number: 95136586
SOMATIC **P53 MUTATIONS** IN HUMAN **BREAST** CARCINOMAS IN AN ICELANDIC POPULATION A **PROGNOSTIC** FACTOR
THORLACIUS S; BORRESEN A-L; EYFJORD J E
MOL. AND CELL BIOL. RES. LAB., ICELANDIC CANCER SOCIETY, SKOGARHID 8, 105 REYKJAVIK, ICELAND.
CANCER RES 53 (7). 1993. 1637-1641. CODEN: CNREA
Full Journal Title: Cancer Research
Language: ENGLISH
Mutations in the p53 gene are among the most common genetic changes in human carcinomas. They have been found in many tumor types including colon, lung, and breast. We have used constant denaturant gel electrophoresis in order to screen samples from 109 breast carcinomas for mutations in four conserved regions, exons 5, 7, and 8, of the p53 gene. Samples were also analyzed for allelic loss of the p53 gene and of markers more distal on chromosome 17p. Mutations were confirmed by DNA sequencing. Mutations were found in 18 of the 109 samples (16.5%). Loss of heterozygosity at 17p was detected in the majority of informative mutated cases. All cases were also screened for germ line mutations, but none were found. The results obtained were analyzed with respect to clinical parameters and prognosis. There was a significant association between p53 mutation and low content of estrogen receptor protein in the tumors (P = 0.01). An association with poor prognosis was strongly indicated by mortality rates that were 37.5% among the patients with p53 mutation and 9.4% for the control group (mean follow up, 32 months). P53 mutation was found to be the strongest negative factor against survival in a covariate survival analysis (P = 0.001).

15/7/11 (Item 11 from file: 5)

DIALOG(R)File 5:BIOSIS PREVIEWS(R)
(c) 1998 BIOSIS. All rts. reserv.

9793329 BIOSIS Number: 44043329
WORSE **PROGNOSIS** AND HIGHER PROLIFERATION RATE IN NODE-NEGATIVE **BREAST** TUMORS WITH **P53 MUTATIONS** IDENTIFIED BY IMMUNOHISTOCHEMISTRY AND SINGLE STRAND CONFORMATIONAL POLYMORPHISM ANALYSIS
ELLEDGE R M; FUQUA S A W; CLARK G M; ALLRED D C
UNIV. TEXAS HEALTH SCI. CENTER, SAN ANTONIO, TEX. 78284.
15TH ANNUAL SAN ANTONIO BREAST CANCER SYMPOSIUM, SAN ANTONIO, TEXAS, USA, DECEMBER 9-10, 1992. BREAST CANCER RES TREAT 23 (1-2). 1992. 132.
CODEN: BCTRD
Language: ENGLISH

15/7/12 (Item 12 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)
(c) 1998 BIOSIS. All rts. reserv.

9617147 BIOSIS Number: 94122147
ACCUMULATION OF P53 TUMOR SUPPRESSOR GENE PROTEIN AN INDEPENDENT MARKER OF PROGNOSIS IN BREAST CANCERS
THOR A D; MOORE D H II; EDGERTON S M; KAWASAKI E S; REIHSAUS E; LYNCH H T ; MARCUS J N; SCHWARTZ L; CHEN L-C; ET AL
DEP. PATHOL., WARREN 2, MASS. GENERAL HOSP., FRUIT ST., BOSTON, MASS. 02114.
J NATL CANCER INST (BETHESDA) 84 (11). 1992. 845-855. CODEN: JNCIE
Full Journal Title: Journal of the National Cancer Institute (Bethesda)
Language: ENGLISH
Background: Mutations of the tumor suppressor gene p53 have been identified in breast cancer cell lines, and some breast carcinomas are detectable by immunohistochemical assay because of p53 protein accumulation. Purpose: This study was designed to determine whether **p53** protein accumulation in **breast** cancers correlates with **p53** gene **mutation**, with survival, and with five pathobiologic factors associated with **prognosis**. Methods: IgG1 monoclonal antibody to human p53 protein (PAb 1801) and immunohistochemical methods were used to detect p53 protein accumulation in archival formalin-fixed, paraffin-embedded, randomly selected carcinomas. We studied 295 invasive ductal carcinomas from the Massachusetts General Hospital; 151 were determined to the sporadic (not hereditary). We also studied 97 invasive ductal carcinomas-21 sporadic and 76 familial (hereditary)-from Creighton University. In addition, we examined 31 archival in situ carcinomas, 15 snap-frozen invasive ductal carcinomas, primary cell cultures from three benign breast tissue samples, and breast carcinoma cell lines MDA-MB-231 and MDA-MB-468. Results: Nuclear p53 protein was observed in 16% of the 31 in situ carcinomas, 22% of the 172 sporadic carcinomas, 34% of the 50 tumors from patients with familial breast cancer, 52% of the 23 tumors from patients with the familial breast and ovarian cancer syndrome, and all three tumors from two patients with the Li-Fraumeni syndrome. There was complete concordance between p53 gene mutation and p53 protein accumulation in the 15 snap-frozen carcinomas and in both breast carcinoma cell lines. Statistically significant associations of p53 protein accumulation with estrogen receptor negativity and with high nuclear grade were found. There were statistically significant associations, independent of other prognostic factors, between p53 protein accumulation and metastasis-free and overall survival, for randomly accrued and for both sporadic and familial tumors. Conclusions: Immunohistochemically detected p53 protein accumulation was an independent marker of shortened survival and was seen more often in familial than in sporadic carcinomas. Our findings also suggest a correlation between p53 protein accumulation and p53 gene mutation.

15/7/13 (Item 13 from file: 5)

DIALOG(R)File 5:BIOSIS PREVIEWS(R)
(c) 1998 BIOSIS. All [redacted]. reserv.

9306757 BIOSIS Number: 43051757
PROGNOSTIC SIGNIFICANCE OF **MUTATIONS** IN THE **P53** GENE IN NODE NEGATIVE **BREAST** CANCER
ELLEDGE R M; FUQUA S A W; CLARK G M; ALLRED D C; MCGUIRE W L
MED./ONCOL., UNIV. TEX. HEALTH SCI. CENT., SAN ANTONIO, TEX. 78284.
83RD ANNUAL MEETING OF THE AMERICAN ASSOCIATION FOR CANCER RESEARCH, SAN DIEGO, CALIFORNIA, USA, MAY 20-23, 1992. PROC AM ASSOC CANCER RES ANNU MEET 33 (0). 1992. 253. CODEN: PAMRE
Language: ENGLISH

15/7/14 (Item 14 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)
(c) 1998 BIOSIS. All rts. reserv.

9081771 BIOSIS Number: 93066771
MUTATIONS IN P53 AS POTENTIAL MOLECULAR MARKERS FOR HUMAN BREAST CANCER
RUNNEBAUM I B; NAGARAJAN M; BOWMAN M; SOTO D; SUKUMAR S
MOLECULAR BIOLOGY BREAST CANCER LABORATORY, SALK INSTITUTE BIOLOGICAL STUDIES, 10010 NORTH TORREY PINES ROAD, LA JOLLA, CALIF. 92037.
PROC NATL ACAD SCI U S A 88 (23). 1991. 10657-10661. CODEN: PNASA
Full Journal Title: Proceedings of the National Academy of Sciences of the United States of America
Language: ENGLISH
Based on the high incidence of loss of heterozygosity for loci on chromosomes 17p in the vicinity of the p53 locus in human breast tumors, we investigated the frequency and effects of mutations in the p53 tumor suppressor gene in mammary neoplasia. We examined the p53 gene in 20 breast cancer cell lines and 59 primary breast tumors. Northern blot analysis, immunoprecipitation, and nucleotide sequencing analysis revealed aberrant mRNA expression, overexpression of protein, and point mutations in the p53 gene in 50% of the cell lines tested. A multiplex PCR assay was developed to search for deletions in the p53 genomic locus. Multiplex PCR of genomic DNA showed that up to 36% of primary tumors contained aberrations in the p53 locus. Mutations in exons 5-9 of the p53 gene were found in 10 out of 59 (17%) of the primary tumors studied by single-stranded conformation polymorphism analysis. We conclude that, compared to amplification of HER2/NEU, MYC, or INT2 oncogene loci, **p53** gene **mutations** and deletions are the most frequently observed genetic change in **breast** cancer related to a single gene. Correlated to disease status, **p53** gene **mutations** could prove to be a valuable marker for **diagnosis** and/or **prognosis** of **breast** neoplasia.

15/7/16 (Item 1 from file: 71)
DIALOG(R)File 71:ELSEVIER BIOBASE
(c) 1998 Elsevier Science B.V. All rts. reserv.

00042709 94093820
p53 gene **mutations** and steroid receptor status in **breast** cancer. Clinicopathologic correlations and **prognostic** assessment
Caleffi M.; Teague M.W.; Jensen R.A.; Vnencak-Jones C.L.; Dupont W.D.; Parl F.F.
ADDRESS: F.F. Parl, United States
Journal: Cancer, 73/8 (2147-2156), **1994**
CODEN: CANCA
ISSN: 0008-543X
DOCUMENT TYPE: Article
LANGUAGES: English SUMMARY LANGUAGES: English

15/7/20 (Item 4 from file: 73)

DIALOG(R)File 73:EMBASE

8513833 EMBASE No: 92189713
p53 Mutations, another **breast** cancer **prognostic** factor
Callahan R.
Division of Cancer Biology, National Cancer Institute, National Institutes of Health, Bethesda, MD 20892 USA
J. NATL. CANCER INST. (USA) , **1992**, 84/11 (826-827)
CODEN: JNCIA ISSN: 0027-8874
LANGUAGES: English

15/7/21 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07759824 94282791
The role of TP53 in breast cancer development.
Eeles RA; Bartkova J; Lane DP; Bartek J
Section of Molecular Carcinogenesis, Institute of Cancer Research, Sutton, Surrey.
Cancer Surv (UNITED STATES) **1993**, 18 p57-75, ISSN 0261-2429
Journal Code: CNG
Languages: ENGLISH
Document type: JOURNAL ARTICLE; REVIEW; REVIEW, ACADEMIC
The TP53 protein is proving to be central to cell cycle control after exposure to DNA damage, and every week a new feature of its role in the regulation of cell division is described. TP53 is the most commonly altered oncogene in human tumours and is involved in the development of both sporadic and some hereditary breast tumours. Although there is no doubt that germline mutations in the TP53 gene carry a high risk of early onset breast cancer, and the gene is mutated somatically in a proportion of preinvasive and invasive breast cancers, it is not usually the initiating genetic event in most breast tumours. It does, however, seem to be an independent prognostic factor for survival and could prove useful in clinical management of node negative breast cancer patients. There has been an explosion of reports about the function of the TP53 protein--in particular, it seems to have a central role in the monitoring of some types of DNA damage. This role may prove to be the most important aspect of its association with breast cancer development, both as a prognostic factor and as a handle for treatment. (115 Refs.)

15/7/22 (Item 2 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07690684 94073808
Overexpression of p53 and prognosis in breast cancer.
Friedrichs K; Gluba S; Eidtmann H; Jonat W
Department of Gynecology and Obstetrics, University of Hamburg, Medical School, Germany.
Cancer (UNITED STATES) Dec 15 **1993**, 72 (12) p3641-7, ISSN 0008-543X Journal Code: CLZ
Languages: ENGLISH
Document type: JOURNAL ARTICLE
BACKGROUND. Assessment of prognostic markers in breast cancer independent of the axillary lymph node status is of major concern for the application of adjuvant treatment regimens. The current treatment decision is based mainly on the axillary lymph node status. Because of improved screening methods, the number and proportion of patients with node-negative disease are increasing, which warrants a search for reliable prognostic parameters. The application of tumor suppressor gene expression appears to be

especially suited as a marker of the progress in malignant cellular dedifferentiation. METHODS. Tumor tissues of 156 patients with primary invasive breast cancer were analyzed immunohistochemically for the presence of p53 protein in paraffin-embedded material. The reaction to monoclonal antibody PAb1801 yielded better results than did reactions to monoclonal antibody DO1 and polyclonal antibody CM-1. The significance of the immunohistochemical data was compared with a panel of established risk factors. RESULTS. Nuclear accumulation of p53 protein proved to be an independent marker of dedifferentiation, regardless of the lymph node status. Tumors showing p53 immunoreactivity were significantly more often related with histological Grade 3 and the absence of steroid hormone receptors. Kaplan-Meier estimation and multivariate analysis of disease-free and overall survival rate corroborated the importance of p53 as a prognostic parameter. CONCLUSION. Overexpression of p53 protein emerged as a reliable and independent predictor for disease recurrence and reduced survival rates in patients with breast cancer.

15/7/23 (Item 3 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07622693 93392918
Relationship between p53 expression and other prognostic factors in human breast carcinoma. An immunohistochemical study.
Martinazzi M; Crivelli F; Zampatti C; Martinazzi S
Postgraduate School of Anatomic Pathology, University of Pavia, Italy.
Am J Clin Pathol (UNITED STATES) Sep **1993**, 100 (3) p213-7,
ISSN 0002-9173 Journal Code: 3FK
Languages: ENGLISH
Document type: JOURNAL ARTICLE
Among 843 cases of breast cancer, p53 oncoprotein was detected by the monoclonal antibody (MoAb) Pab-1801 in only 13%. Low-grade carcinomas (tubular, mucinous, papillary, and invasive cribriform types) did not express p53 protein, but it was observed in 4.2% of infiltrating lobular carcinomas (6 of 140 cases) and 50% of pure medullary carcinomas (5 of 10 cases). In intermediate-grade neoplasms, no correlation was seen between p53 status and other putative determinants of a poor prognosis. The latter included high tumor stage, lymph nodal involvement, high growth fraction (as determined by labeling with the MoAb Ki-67), negative results for estrogen receptor (ER) and progesterone receptor (PR) proteins, and amplification of the c-erbB-2 oncogene product in the neoplastic cells. Ninety-nine of 640 (15.5%) cases of high-grade, invasive, ductal breast carcinoma, however, showed an inverse relationship between expression of p53 protein and positive results for ER/PR proteins and a direct correlation with large tumor size, Ki-67-determined growth fraction, and amplification of c-erbB-2 oncopeptide. All of the latter associations were highly significant statistically. The authors conclude that mutant p53 protein may serve a prognostic role in a subset of cases of invasive ductal mammary carcinoma.

15/7/24 (Item 4 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

07352687 94155285
The p53 tumor suppressor gene. A preliminary clinical study in breast cancer patients.
Micelli G; Donadeo A; Quaranta M
Clinical-Chemistry Laboratory, Oncology Institute, Bari, Italy.
Cell Biophys (UNITED STATES) Aug-Dec **1992**, 21 (1-3) p25-31,
ISSN 0163-4992 Journal Code: CQC
Languages: ENGLISH
Document type: JOURNAL ARTICLE

p53 was originally considered to be a nuclear oncogene, but several convergent lines of research have indicated that the wild-type gene functions as a tumor suppressor gene negatively regulating the cell cycle. Mutations in the p53 gene have been detected in many tumor types and seem to be the most common genetic alterations in human cancer. In this preliminary study, sera of 92 patients (pts) with breast disease were analyzed for the presence of the mutant p53 protein (mp53) with a selective immunoenzyme assay employing a monoclonal antibody (PAb 240) specific for the majority of mammalian m p53 but not for the wild-type protein. Of the 10 patients with benign breast disease, only two (20%) showed detectable m p53 levels in the serum. In the breast cancer group, sera from 7 of the 30 pts (23%) without lymph node involvement were positive for m p53, as were 7 out of the 45 pts (15%) with metastatic lymph nodes and 1 out of the 7 pts (14%) with disseminated disease. The specificity of m p53 assay evaluated in 20 healthy controls was 100%. These preliminary results showed that serum positivity for m p53 is not related to breast disease extension. Further studies to assess the utility of m p53 as a possible prognosis factor in breast cancer are currently in progress.

15/7/28 (Item 2 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

120214008 CA: 120(17)214008d JOURNAL
The role and prognostic significance of p53 gene alterations in breast cancer
AUTHOR(S): Elledge, Richard M.; Fuqua, Suzanne A. W.; Clark, Gary M.; Pujol, Pascal; Allred, D. Craig
LOCATION: Health Sci. Cent., Univ. Texas, San Antonio, TX, USA
JOURNAL: Breast Cancer Res. Treat. DATE: 1993 VOLUME: 27 NUMBER: 1-2
PAGES: 95-102 CODEN: BCTRD6 ISSN: 0167-6806 LANGUAGE: English
SECTION:
CA214001 Mammalian Pathological Biochemistry
CA203XXX Biochemical Genetics
IDENTIFIERS: gene p53 mutation breast cancer prognosis
DESCRIPTORS:
Mammary gland,neoplasm...
gene p53 mutations in, of humans, prognosis in relation to Mutation...
in gene p53, in breast cancer, of humans, prognosis in relation to Gene,animal, TP53...
mutations in, in breast cancer, of humans, prognosis in relation to

15/7/29 (Item 3 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

120213913 CA: 120(17)213913h JOURNAL
Prognostic significance of TP53 alterations in breast carcinoma
AUTHOR(S): Andersen, T.I.; Holm, R.; Nesland, J.M.; Heimdal, K.R.; Ottestad, L.; Boerresen, A. L.
LOCATION: Dep. Genet., Norw. Radium Hosp., N-0310, Oslo, Norway
JOURNAL: Br. J. Cancer DATE: 1993 VOLUME: 68 NUMBER: 3 PAGES: 540-8
CODEN: BJCAAI ISSN: 0007-0920 LANGUAGE: English
SECTION:
CA214001 Mammalian Pathological Biochemistry
IDENTIFIERS: gene TP53 mutation breast carcinoma human, antigen p53 mutation breast carcinoma human
DESCRIPTORS:
Gene,animal, c-erbB2...
amplification of, in human breast carcinoma, gene TP53 mutations in relation to
Mammary gland,neoplasm, carcinoma...

gene TP53 mutations in, in human, prognostic significance of
Mutation,deletion... [redacted]tation,point... Mutation,trans[redacted]on...
in gene TP53 in [redacted]an breast carcinoma, prognostic significance of
Estrogens,receptors... Progestogens,receptors... Receptors,estrogen...
Receptors,progestogen...
in human breast carcinoma, gene TP53 mutations in relation to
Gene,animal, TP53... Phosphoproteins,tumor suppressor, p53...
mutations in, in human breast carcinoma, prognostic significance of
Deoxyribonucleic acid sequences...
of gene TP53 mutations, in human breast carcinoma, prognostic
significance of
Protein sequences...
of tumor antigen p53 mutations, in human breast carcinoma, prognostic
significance of
Cell nucleus...
tumor antigen p53 accumulation in, in human breast carcinoma, gene
mutation in relation to
Genetic element,exon...
7, of gene TP53 in human breast carcinoma, prognostic significance of
mutations in

17/7/1 (Item 1 from file: 357)
DIALOG(R)File 357:Derwent Biotechnology Abs

193793 DBA Accession No.: 96-05200 PATENT
Sequence-based diagnosis of a human neoplastic tissue, blood or body fluid - cancer diagnosis by DNA amplification and DNA sequencing using a DNA primer set specific for **p53** tumor suppressor, WAFI, erbB2 (HerII/Neu), p16 (MTS-I), MTS-II, MLH-1, MLH-2 or Ras gene
AUTHOR: **Bywater M**; Lindstrom P; Inganas M
CORPORATE SOURCE: Uppsala, Sweden.
PATENT ASSIGNEE: Pharmacia-Biotech 1996
PATENT NUMBER: WO 9602671 PATENT DATE: 960201 WPI ACCESSION NO.: 96-105932 (9611)
PRIORITY APPLIC. NO.: SE 943953 APPLIC. DATE: 941116
NATIONAL APPLIC. NO.: WO 95SE804 APPLIC. DATE: 950629
LANGUAGE: English
ABSTRACT: A new method for cancer diagnosis (e.g. lung carcinoma, prostate carcinoma, stomach carcinoma, colorectal carcinoma, melanoma, leukemia or preferably mamma carcinoma) involves detection of a mutation in genomic DNA or cDNA encoding a cancer-related protein, determining the presence, nature and location of the mutation and influence on biological function (e.g. biological aggressiveness and/or metastasis potential of the tumor), and forming a prognosis with guidance for therapy. A new oligonucleotide DNA primer set may be used for DNA amplification and/or DNA sequencing of **p53** tumor suppressor genomic DNA or cDNA. The mutation may be in a DNA binding domain and/or transactivating site, especially in an evolutionarily conserved region. The gene is preferably a WAFI, erbB2 (HerII/Neu), p16 (MTS-I), MTS-II, MLH-1, MLH-2, Ras or (preferably) **p53** gene. The gene is amplified, sequenced and products are detected in an automated sequencer, with optional computer software to track samples and control process steps. The method is more reliable than previous methods, and

22/3/1 (Item 1 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

14180370 BIOSIS Number: 01180370
Assessment of sequence-based **p53** gene analysis in human breast cancer: Messenger RNA in comparison with genomic DNA targets
Williams C; Norberg T; Ahmadian A; Ponten F; Bergh J; **Inganas M**; Lundeberg J; Uhlen M
Dep. Biochem. and Biotechnol., Royal Inst. Technol., S-100 44 Stockholm, Sweden
Clinical Chemistry 44 (3). 1998. 455-462.
Full Journal Title: Clinical Chemistry
ISSN: 0009-9147
Language: ENGLISH
Print Number: Biological Abstracts Vol. 105 Iss. 009 Ref. 123082

22/3/5 (Item 5 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

13613750 BIOSIS Number: 99613750
The **p53** gene in breast cancer: Prognostic value of complementary DNA sequencing versus immunohistochemistry
Sjogren S; **Inganas M**; Norberg T; Lindgren A; Nordgren H; Holmberg L ; Bergh J
Dep. Oncol., Univ. Uppsala, Akademiska Sjukhuset, S-751 85 Uppsala, Sweden
Journal of the National Cancer Institute (Bethesda) 88 (3-4). 1996. 173-182.
Full Journal Title: Journal of the National Cancer Institute (Bethesda)
ISSN: 0027-8874
Language: ENGLISH
Print Number: Biological Abstracts Vol. 104 Iss. 003 Ref. 039764

22/3/6 (Item 6 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

13402465 BIOSIS Number: 99402465
Prognostic value of sequenced based and immunohistochemistry diagnosis of the **p53** gene in lymph node negative breast cancer patients
Wilking N; Rutqvist L E; Skoog L; **Inganas M**; Stockholm Breast Cancer Study Group
Dep. Oncol., Karolinska Hosp., Stockholm, Sweden
Breast Cancer Research and Treatment 41 (3). 1996. 252.
Full Journal Title: 19th Annual San Antonio Breast Cancer Symposium on Breast Cancer Research and Treatment, San Antonio, Texas, USA, December 11-14, 1996. Breast Cancer Research and Treatment
ISSN: 0167-6806
Language: ENGLISH
Document Type: CONFERENCE PAPER
Print Number: Biological Abstracts/RRM Vol. 049 Iss. 003 Ref. 049104

22/3/7 (Item 7 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

12154714 BIOSIS Number: 98754714
Mutation analysis of the **p53** gene using cDNA based sequencing is superior to immunohistochemical analysis with Pab 1801 on a consecutive and population based primary breast cancer material regarding prognostic information and response to adjuvant therapy
Bergh J; Sjogren S; Norberg T; Jansson T; Nordgren H; Lindgren A; **Inganas M**; Holmberg L
Dep. Oncology, Uppsala Univ., Uppsala, Sweden
Breast Cancer Research and Treatment 37 (SUPPL.). 1996. 39.
Full Journal Title: 18th Annual San Antonio Breast Cancer Symposium, San Antonio, Texas, USA, December 8-13, 1995. Breast Cancer Research and Treatment
ISSN: 0167-6806
Language: ENGLISH
Document Type: CONFERENCE PAPER

Print Number: Biological Abstracts/RRM Vol. 048 Iss. 005 Ref. 071503

22/3/8 (Item 8 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

11995878 BIOSIS Number: 98595878
P53 Status predicts survival in breast cancer patients treated with or without postoperative radiotherapy: A novel hypothesis based on clinical findings
Jansson T; **Inganas M**; Sjogren S; Norberg T; Lindgren A; Holmberg L; Bergh J
Dep. Oncol., Univ. Uppsala, Akademiska sjukhuset, SE-751 85 Uppsala, Sweden
Journal of Clinical Oncology 13 (11). 1995. 2745-2751.
Full Journal Title: Journal of Clinical Oncology
ISSN: 0732-183X
Language: ENGLISH
Print Number: Biological Abstracts Vol. 101 Iss. 002 Ref. 023583

22/3/9 (Item 9 from file: 5)
DIALOG(R)File 5:BIOSIS PREVIEWS(R)

11748295 BIOSIS Number: 98348295
Evaluation of a large number of breast tumor samples processed by automatic DNA sequencing of the **p53** gene
Sevigny P; Bjorkesten L; Pernemalm P-A; **Inganas M**; Andell Y; Eriksson S; Norberg T
Pharmacia Biotech AB, Uppsala, Sweden
Clinical Chemistry 41 (S6 PART 2). 1995. S221.
Full Journal Title: 47th Annual Meeting of the American Association for Clinical Chemistry, Inc., Anaheim, California, USA, July 16-20, 1995. Clinical Chemistry
ISSN: 0009-9147
Language: ENGLISH
Document Type: CONFERENCE PAPER
Print Number: Biological Abstracts/RRM Vol. 047 Iss. 008 Ref. 140755

22/3/10 (Item 1 from file: 155)
DIALOG(R)File 155:MEDLINE(R)

09468323 98196537
Prognostic value of **P53** gene mutations in a large series of node-negative breast cancer patients.
Falette N; Paperin MP; Treilleux I; Gratadour AC; Peloux N; Mignotte H; Tooke N; Lofman E; **Inganas M**; Bremond A; Ozturk M; Puisieux A
Unite d'Oncologie Moleculaire, Unite INSERM U453, Centre Leon Berard, Lyon, France.
Cancer Res (UNITED STATES) Apr 1 1998, 58 (7) p1451-5, ISSN 0008-5472 Journal Code: CNF
Languages: ENGLISH
Document type: JOURNAL ARTICLE

22/3/11 (Item 1 from file: 357)
DIALOG(R)File 357:Derwent Biotechnology Abs

193793 DBA Accession No.: 96-05200 PATENT
Sequence-based diagnosis of a human neoplastic tissue, blood or body fluid

- cancer diagnosis by DNA amplification and DNA sequencing using a DNA primer set speci[redacted] for **p53** tumor suppressor, WAF[redacted]erbB2 (HerII/Neu), p16 (MTS-I), MTS-II, MLH-1, MLH-2 or Ras gene
AUTHOR: Bywater M; Lindstrom P; **Inganas M**
CORPORATE SOURCE: Uppsala, Sweden.
PATENT ASSIGNEE: Pharmacia-Biotech 1996
PATENT NUMBER: WO 9602671 PATENT DATE: 960201 WPI ACCESSION NO.: 96-105932 (9611)
PRIORITY APPLIC. NO.: SE 943953 APPLIC. DATE: 941116
NATIONAL APPLIC. NO.: WO 95SE804 APPLIC. DATE: 950629
LANGUAGE: English

22/3/12 (Item 2 from file: 357)
DIALOG(R)File 357:Derwent Biotechnology Abs

177800 DBA Accession No.: 95-04621
A new approach in automated DNA sequencing to analyze the **p53** gene in a large number of breast cancer patients - diagnosis by mamma carcinoma automated DNA sequencing and large-scale polymerase chain reaction (conference abstract)
AUTHOR: Andell Y; Eriksson S; **Inganas M**; Norberg T; Sevigny P
CORPORATE AFFILIATE: Pharmacia-Biotech
CORPORATE SOURCE: Pharmacia Biotech AB, Uppsala, Sweden.
JOURNAL: Clin.Chem. (40, 12, 2337) 1994
ISSN: 0009-9147 CODEN: CLCHAU
CONFERENCE PROCEEDINGS: The Genetic Revolution, San Diego, California, 17-19 November, 1994.